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INVITED REVIEW

PYOGENIC ABSCESSES AND PARASITIC DISEASES

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SUMMARY

Parasitic diseases which during their course in the host switch the immune system from a T helper 1 to a T helper 2 response may be detrimental to the host, contributing to granuloma formation, eosinophilia, hyper-IgE, and increased susceptibility to bacterial and fungal infections. Patients and animals with acute schistosomiasis and hyper-IgE in their serum develop pyogenic liver abscess in the presence of bacteremia caused by *Staphylococcus aureus*. The *Salmonella-S. mansoni* association has also been well documented. The association of tropical pyomyositis (pyogenic muscle abscess) and pyogenic liver abscess with *Toxocara* infection has recently been described in the same context. In tropical countries that may be an interesting explanation for the great morbidity of bacterial diseases. If the association of parasitic infections and pyogenic abscesses and/or fungal diseases are confirmed, there will be a strong case in favor of universal treatment for parasitic diseases to prevent or decrease the morbidity of superinfection with bacteria and fungi.

KEYWORDS: Liver abscess; Pyomyositis; Schistosomiasis; Toxocariasis; Th1/Th2 paradigm; Staphylococci.

INTRODUCTION

Some parasitic diseases may have a pivotal role in the development of bacterial infections and/or aggravation of other infectious diseases. Herein our experience and arguments in favor of an association of pyogenic abscesses and parasitic diseases are reviewed.

TH1/TH2 PARADIGM, ALLERGY, HYPER-IgE AND MIGRATING LARVAE OF WORMS

The T helper (Th1) and Th2 patterns of cytokine were originally described among mouse CD4+ T-cell clones and later among human T cells. Mouse Th1 cells produce interleukin 2 (IL-2), interferon γ (IFN-γ) and lymphotoxin (LT), whereas Th2 cells produce IL-4, IL-5, IL-6, IL-9, IL-10 and IL-13 and is not as tighly restricted to a single subset as in mouse T cells[29].

A logical functional classification of T-cell subsets must be based on the known biological activities of the cytokines these T cells produce (Fig. 1). The principal Th1 effector cytokine, IFN-γ, has two key functions. First, it activates macrophages, enhancing their microbicidal actions. Second, IFN-γ stimulates the production of IgG antibodies which bind to high-affinity Fcγ receptors and complement proteins and are therefore the principal antibodies involved in the opsonization and phagocytosis of particulate microbes. Th1-dominant immune responses are often associated with inflammation and tissue injury, because two Th1 cytokines, TNF-ß and IFN-γ, recruit and activate inflammatory leukocytes. The typical inflammatory reaction is delayed type hypersensitivity (DTH), and is often a price that is paid for protective immunity against microbes such as mycobacteria. Some Th1 cells acquire cytolytic capacity, and the cytokines produced by Th1 cells, notably IL-2 and IFN-γ, promote the differentiation of CD8+ T lymphocytes into active cytotoxic cells. These are the other ways in which the Th1 subset participates in the elimination of intracellular microbes.

The signature of cytokines of Th2 cells are IL-4 and IL-5. IL-4 is the major inducer of B-cell switching to IgE production and is therefore a key initiator of IgE-dependent, mast-cell-mediated reactions. IL-5 is the principal eosinophil-activating cytokine, and mice lacking IL-5 or its receptor show marked defects in eosinophil responses to helminths[2]. The production of these two cytokines by the same subset accounts for the frequent presence of both IgE and activated eosinophils in Th2-dominated immune reactions, such as in allergies and helminthic infections. The recruitment of eosinophils to sites of immune reactions is mediated by chemokines, including eotaxin, which are produced by both Th1 and Th2 subsets, and by non-T cells, and the activity of both is augmented by local IL-4 and IL-5 production. It is noteworthy that several cytokines produced by Th2 cells have anti-inflammatory actions. IL-4

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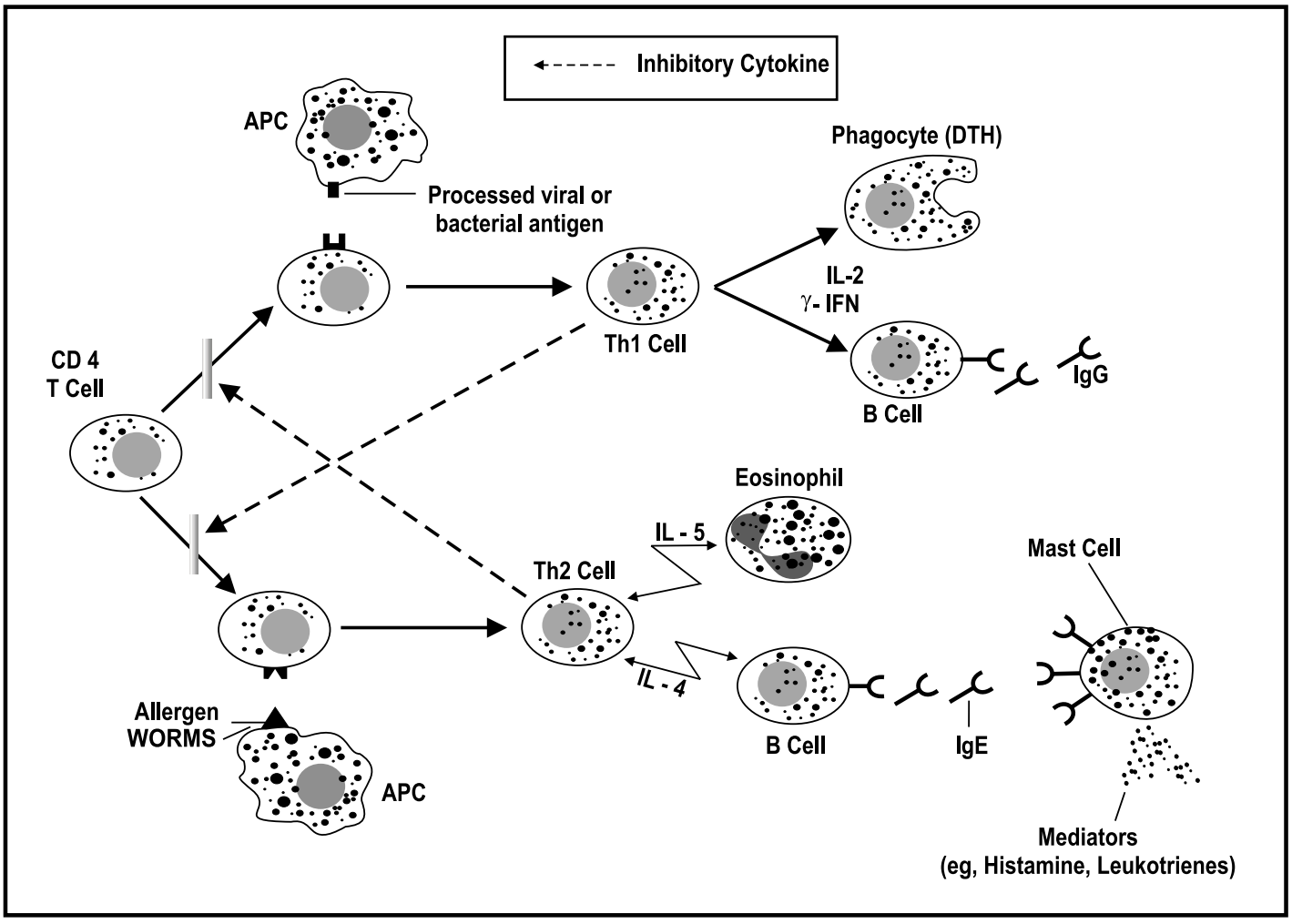

Fig. 1 - The delicate balance between the Th1/Th2 pattern of immune response. In allergic individuals and in helminthic infections there is a Th2-dominated immune reaction with eosinophilia, and hyper-IgE; by binding to mast cells, IgE triggers release of mediators associated with pruritus. When activated, Th2 cytokines inhibit the Th1 arm of the immune system. APC = antigen presenting cell; DTH = delayed type hypersensitivity.

and IL-13 antagonize the macrophage-activating action of IFN-γ, IL-10 suppresses numerous macrophage responses, and TGF-ß is antiproliferative and inhibits leukocyte activation. Thus, the net result of Th2 activation is to inhibit acute and chronic inflammation, including DTH reactions. This raises the possibility that an important physiological function of Th2 is not as effectors but as regulators of immune responses[2,15,29].

The balance between Th1 and Th2 responses to an infectious agent can influence both pathogen growth and immunopathology. It is now appreciated that resistance to many intracellular microbes, including bacteria, protozoa and fungi, is linked to the induction of Th1 responses, and, in particular, with macrophage-activating cytokines, IFN-γ and TNF-α. In contrast to intracellular microbes, extracellular pathogens and particularly helminths typically trigger Th2-dominated responses. It is also possible that helminths have evolved the capacity to induce Th2 responses to protect themselves against potentially toxic Th1-dependent antiparasite effector mechanisms. Regardless of the mechanisms and protective value of antihelminthic Th2 responses, it is clear that such responses may also be detrimental to the host, contributing to granuloma formation, hypereosinophilia and superinfection with bacteria and/or fungi.

The cytokines secreted by each helper T cell subpopulation exert positive feedback on their own subpopulation while inhibiting the opposing subpopulation (Fig. 1).

Allergic reactions involving IgE and mast cells are due to the development and activation of allergen-specific Th2 cells. Individuals with severe atopy have high levels of total and allergen-specific serum IgE[15].

The hyper-IgE syndrome as originally described by BUCKLEY *et al* in 1972 consisted of undue susceptibility to infection, particularly with staphylococci, and extremely high serum IgE levels (> 2000 ng/ml)[5]. It was subsequently shown that patients with this disorder also have a neutrophil granulocyte chemotactic defect. Since then a variety of allied

syndromes have been recorded in which the common feature is the high serum IgE level frequently in association with atopic disorders. The pathogenetic basis for the neutrophil chemotactic defect in the hyper-IgE syndrome has not been established but it has been suggested that it may be due to an inhibitory effect of histamine on neutrophil chemotaxis.

The association of infection and atopy has been the subject of many investigations. Clearly, patients with atopic dermatitis have high rates of colonizations by pathogenic bacteria. For example, *Staphylococcus aureus*, found in 5% of normal control subjects, can be isolated from affected skin in more than 90% of atopic dermatitis patients[4,12].

Parasitic diseases show some features of allergic flares. In acute schistosomiasis there is a hypersensitivity state heralded by itchy cutaneous manifestations (cercarial dermatitis, angioedema, urticaria, bronchospasm) and laboratory data (eosinophilia and high serum IgE levels), among others. Such findings have also been reported for migrating larvae of other parasites being the *Toxocara* infection a good example[6,7,9].

PYOGENIC LIVER ABSCESSES AND SCHISTOSOMIASIS

Bacterial abscesses of the liver are relatively uncommon in developed countries, despite the frequency of cholecystitis, appendicitis and diverticulitis, which are frequently sources of bacterial infection in the liver. Also, in developed countries hepatic abscesses are rare in children but, when present, they occur up to 5 years of age and can be associated with thrombophlebitis of the umbilical vein, abdominal traumatism and immunosuppressive disease.

Pyogenic hepatic abscess is frequently polymicrobial. Enteric Gram-negative bacilli, usually *Escherichia coli*, have been cultured from the majority of pyogenic hepatic abscesses. The specific types of microorganisms that cause hepatic abscess probably vary with the underlying disease. *Staphylococcus aureus* abscesses in the liver may be associated with microabscesses in other organs as part of generalized haematogenous dissemination in children with impaired host defenses (e.g., in acute leukemia).

In the past, it was believed that about 10-20% of amoebic abscesses were secondarily infected with bacteria, usually of enteric origin. However, in a more recent series, superinfection was found to have occurred only in 0 to 4% of cases[3].

We have noticed regional differences in the clinical presentation of liver abscesses in South America when compared to northern countries[13] . Nowadays, in developed countries, pyogenic liver abscesses are more often described in individuals of middle age or older, especially those who have underlying biliary tract diseases with obstruction and bile stasis that favor infection. Before antibiotics, appendicitis with bacterial seeding of the liver through the portal vein was a frequent cause. Pyogenic liver abscesses secondary to intestinal diseases are well recognized in patients with a variety of disorders including Crohn's disease and diverticulitis. Penetrating wounds to the liver may also lead to abscesses.

In some areas of Brazil, however, pyogenic liver abscesses have been more frequently observed in young individuals (under 15 years of age), and *S. aureus* has been the main bacteria cultured of aspirates from the abscesses[17,18,22]. Acute schistosomiasis and migrating larvae of other parasitic diseases (*Ascaris, Strongyloides, Toxocara*) have been implicated as facilitating factors in the genesis of pyogenic abscesses and staphylococcal infection[33].

Some mechanisms have been proposed as a probable explanation for the association of schistosomiasis and pyogenic liver abscesses[17,18,21,22,24,25]: (1) liver necrosis caused by eggs or dead worms of *S. mansoni* could be colonized by bacteria; (2) there is transient impairment of the cell mediated immunity in the acute phase of schistosomiasis in animal models; (3) the literature contains several reports of cases of recurrent infections caused by *S. aureus* in the presence of high IgE levels in the serum, and high serum IgE levels have been reported in acute schistosomiasis.

***Case report*:** An 8 year-old boy who complained of abdominal pain associated with decreased activity, fever and vomiting starting 12 days before was admitted to hospital. His axillary temperature was 39 °C and he appeared acutely ill. Respirations were laboured and the respiratory rate was 32/min. His abdomen was distended and painful to palpation. Initial laboratory tests showed anemia, leukocytosis and eosinophilia. Liver function tests were normal. Three blood cultures grew no organisms and stool examination revealed viable eggs of *Schistosoma mansoni*. A chest X-ray showed opacification of the right costophrenic angle.The patient received clindamycin, oxacilin and gentamycin for 7 days without improvement. Ultrasonography and a CT scan of the liver revealed the presence of one large abscess surrounded by 3 small satellite abscesses (Fig. 2). During laparotomy the large abscess was drained (*Staphylococcus aureus* was the sole bacterium isolated from the purulent material obtained), and liver biopsies were performed at 2 different sites. A diagnosis of acute schistosomiasis was confirmed by the identification of a miliary distribution of necrotic-exudative granulomata around eggs of *Schistosoma mansoni* in the liver. In addition to antibiotics the patient was treated with oxamniquine and corticosteroids and made an uneventful recovery.

***Other cases*:** The medical charts of 50 patients with pyogenic liver abscesses who were admitted to 3 general hospitals of Belo Horizonte, state of Minas Gerais, in Brazil, during a 10 years' period, were reviewed retrospectively by LAMBERTUCCI *et al.*[22]. Demographic information, chief complaints, clinical and laboratory findings, and response to treatment were recorded. The age range varied from 2 to 79 years (41% were younger than 15). The main presenting complaints were fever and non-specific abdominal pain. Eosinophilia was observed in 15 cases (30%). Parasitological stool examination was obtained from 28 patients with liver abscesses, and in 20 (71%) the following agents were diagnosed in the stools: *S. mansoni* (36%), *Strongyloides stercoralis* (21%), *Ascaris lumbricoides* (18%), and *Entamoeba histolytica* (4%). Ultrasonography had a major role in the diagnosis of liver abscesses and in treatment, directing the percutaneous drainage of the purulent collections. Culture of the material obtained during surgery or by percutaneous puncture in 23 patients revealed: *S. aureus* (14 cases), *S. viridans* (2 cases), Gram-negative bacteria (5 cases), the association of Gram-positives and Gram-negatives (2 cases). Two out of the 5 patients submitted to liver biopsy had the necrotic-exudative granulomata of *Schistosoma mansoni* identified in the liver.

***Experimental studies*:** Male albino mice were infected with 40 *Schistosoma mansoni* cercariae of the LE strain. A strain of coagulase positive *Staphylococcus aureus* that had been isolated from a patient with staphylococcal bacteremia was cultivated for 24 h at 37 °C on

nutrient agar. After incubation, the bacteria were resuspended in 0.85% saline. Dilutions were made in saline and the number of organisms counted as plaques. Each mouse was injected intravenously with 0.8 x 10^6 to 4 x 10^6 units per infection[39].

Mice were divided into four groups as follows. Group 1, 17 mice infected with *S. mansoni* and *S. aureus*; group 2, 17 mice infected with *S. mansoni* only; group 3, 19 mice infected with *S. aureus* only; group 4, 18 uninfected mice kept as controls. All mice were killed 30 days after inoculation with the bacteria. The abdominal organs were carefully examined and blood was collected for culture. Purulent material from the abscesses was also cultured.

Thirteen of 17 group 1 mice (77%) developed multiple hepatic abscesses. Microscopy of the liver showed multiple schistosome granulomata and several abscesses, of variable size, consisting of a dense polymorphonuclear neutrophil exudate, necrotic material, and many colonies of Gram-positive cocci. In some mice the colonies were found surrounding *S. mansoni* adult worms (Fig. 3). The 4 remaining group 1 mice and mice from group 2 showed only granulomas of schistosomiasis in their livers. No pathological change was observed by microscopy in the livers of mice of groups 3 and 4. *S. aureus* was recovered from the blood of 4 group 1 mice and 3 group 3 mice. This was the only bacterium recovered from the multiple hepatic abscesses of group 1 mice.

THE ASSOCIATION OF SCHISTOSOMA AND ENTEROBACTERIACAE

Chronic persistent *Salmonella* bacteremia has been described in association with *S. mansoni* infection[30]. The most common characteristics of the clinical syndrome are: (1) an indolent febrile disease; (2) bacteremia with one of many species of the genus *Salmonella*; (3) chronic active schistosomiasis. The clinical features of this peculiar association have been described as being more like kalazar than typhoid fever[23]. When diagnosed correctly, mortality is low and the response to antibiotic therapy, dramatic. However, recurrent *Salmonella* bacteremia is common if the underlying schistosome disease is not treated.

The association of *S. mansoni* with *Escherichia coli* has also been described[10,38]. After months of febrile attacks, treatment with co-trimoxazole or amoxicillin followed by antischistosomal drugs resulted in cure. ROCHA *et al*.[37] inoculated *Escherichia coli* into mice previously infected with *Schistosoma mansoni* and observed bacteremia and liver abscesses in the experimental group. The formation of abscesses was interpreted on the occasion as being derived from suppurative cholangitis.

The number of cases of bacteremia due to Gram negative bacteria other than *Salmonellae* should not increase since it is known that most Gram negatives parasitize and kill the schistosomes[31].

LAMBERTUCCI *et al*.[19] described the cases of 2 young men with *Salmonella* bacteremia, active schistosomiasis and the acquired immunodeficiency syndrome. The clinical presentation comprised nonspecific signs and symptoms, such as fatigue, malaise, weight loss, diarrhea, prolonged fever, and hepatosplenomegaly. In one patient, liver biopsy showed poorly formed granulomata around *S. mansoni* eggs and hepatitis. Treatment of schistosomiasis alone induced consistent clinical improvement with eventual cure of both *Salmonella* and *S. mansoni* infections. Recognition of the *Salmonella-S. mansoni* association in patients with AIDS is important because treatment of schistosomiasis makes a difference, improving the prognosis of this otherwise, recurrent, potentially fatal bacteremia.

TROPICAL PYOMYOSITIS AND TOXOCARIASIS

Pyomyositis (primary muscle abscess) is an acute bacterial infection of skeletal muscle usually caused by *Staphylococcus aureus*. Most cases of pyomyositis occur in the tropics; thence, the term tropical pyomyositis. It accounts for 1 to 4% of hospital admissions in some tropical areas. Bacterial invasion of muscle is assumed to occur after transient bacteremia which leads to seeding of a site of prior muscle damage. It occurs at all ages, in the tropics more frequently among children. As yet, no convincing evidence to relate pyomyositis causally to predisposing circumstances peculiar to the tropics (e.g., malaria, filariasis, arbovirus infection) has been developed.

Skeletal muscle is usually resistant to infection. As an example, abscess formation in the muscle of dogs is inducible after intravenous injection of *S. aureus* only if the muscle is initially traumatized by pinching, electric shock, or ischemia. However, only 25 to 50 percent of humans with pyomyositis report a history of trauma. A number of other predisposing factors have also been identified: (1) Polymorphonuclear leukocytes (PMNs) play an important role in host defenses against staphylococci. Thus, neutropenia or qualitative defects of neutrophil function, such as abnormalities in chemotaxis, ingestion, or bactericidal activity, can predispose to pyomyositis; (2) A local IgE response to certain parasitic infections may secondarily impair neutrophil activity. Alternatively, the increased serum IgE concentration may not be the primary cause of PMN dysfunction, but may reflect underlying immune dysregulation.

Clinically, pyomyositis is characterized by fever, localized muscle pain and stiffness, swelling, and tenderness. The definitive diagnosis should be made by aspiration or surgical drainage of the abscess. Aspiration may be facilitated by localizing the site of muscle involvement by ultrasound or CT scan. Gallium scan can be useful in securing the diagnosis or in localizing an occult abscess. Magnetic resonance imaging may be better than computerized tomography for early detection of pyomyositis.

Toxocara canis, the dog *Ascaris*, can infect noncompatible hosts such as humans and remain in their organisms as migrating larvae arrested in their development but metabolically active, eliciting mechanical damages that are exacerbated by host's immune response. Larvae migrate through the tissues of many organs like the liver, lungs, brain, eyes, kidneys, muscles, spinal cord, among others. They frequently induce the formation of granuloma that can be the site of bacterial colonization especially with *Staphylococcus aureus* [34, 35].

Case report: A 16 year-old boy was admitted to hospital with a history of fever, generalized myalgia, diffuse erythema and edema of the lower limbs that started 14 days before. At admission he looked ill, febrile, with generalized erythema, and swelling in the right thigh, right arm and left thigh and gluteous (Fig. 4). He was treated with oxacillin and also submitted to surgical drainage of great quantity of purulent material and necrotic tissue in the affected areas. His condition deteriorated notwithstanding, as he developed respiratory failure

secondary to pneumonia. At this time, a blood examination revealed leukocytosis of 28,000 cells and eosinophilia of 10,000/mm^3. Culture of the abscesses showed *Staphylococcus aureus* resistant to penicillin. He was treated with antibiotics, parenteral nutrition and was eventually discharged from hospital in good condition. A laboratory make up showed serum IgE of > 2,000 KU/L (normal value: < 140 KU/L), and an ELISA for toxocariasis of 1:1042 (positive values: > 1:500). He tested HIV negative. In the follow up visits in the outpatient clinic progressive improvement was observed but he was left with extensive scars in the areas affected.

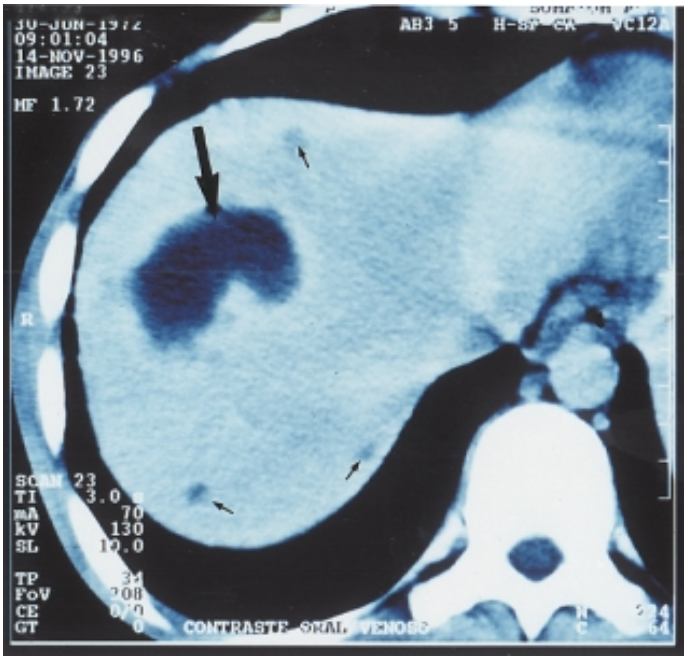

Fig. 2 - Computerized tomography of the liver showing a large abscess (thick arrow) and 3 small satellite abscesses (thin arrows).

Studies in humans and in animals: Between March 1995 and March 1998, we prospectively studied 20 patients with tropical pyomyositis who were admitted to 5 hospitals in Belo Horizonte, Brazil, and who were referred to us for treatment. We enrolled a control group of 20 patients matched by age (± 2 years) and sex who were admitted to the same hospitals. Demographic characteristics of the control group were similar to those of the study group. Serological tests for toxocariasis were performed by ELISA[8,11]. Serology for toxocariasis was positive in 8 (40%) of the 20 patients and 1 (5%) of the controls ($p = 0.008$)[36].

An experimental study was also set up. Fifty-eight Swiss mice, weighing 25 to 30 g, were divided into four groups: 29 mice were infected with 1000 embryonated eggs of *Toxocara canis* at day 0 and weekly with *S. aureus*; 13 mice were infected with only *S. aureus*; 6 mice infected only with 1000 infective eggs of *T. canis* at day 0; and 10 served as a control group without any infection. Infection with larva was undertaken according to the technique of ABO-SHEHADA *et al*.[1]. For infection with *S. aureus*, bacteria were isolated from patients admitted to the hospital with staphylococcal infections; mice were injected via a caudal vein. The number of bacteria varied from 0.8×10^6 to 4×10^6 units per infection. Mice were observed daily and sacrificed when they became ill, or examined postmortem when they died. The carcasses were examined for the presence of muscle abscesses. Specimens were taken from areas with abscess and from apparently normal muscles and were kept in 10% formalin until processed for standard histological study. Muscle abscesses were more frequently observed in mice infected with *T. canis* and *S. aureus* ($p < 0.01$). *S. aureus* was isolated from abscesses of 3 mice. On microscopic examination, a constant feature was the presence of a collection of purulent material, often associated with

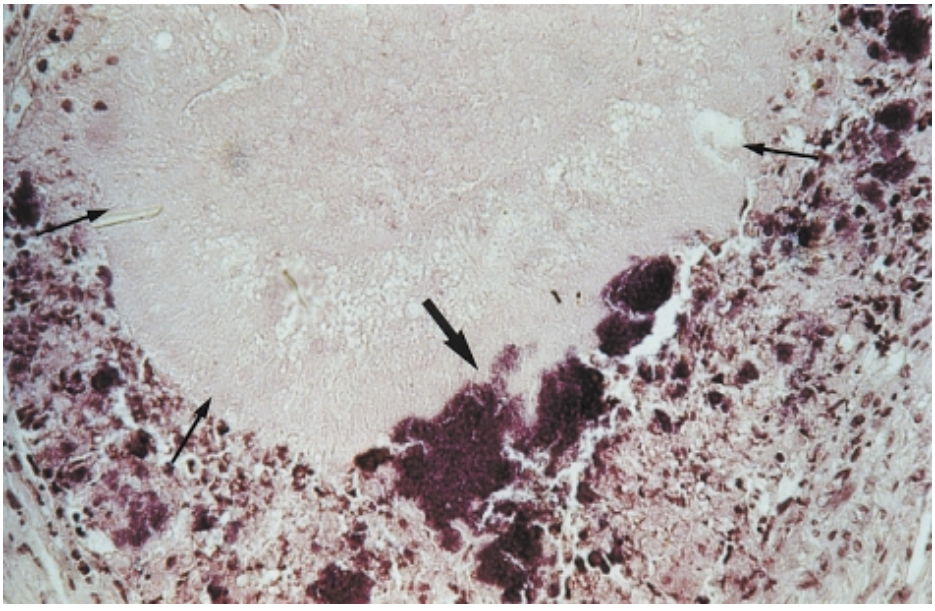

Fig. 3 - A dead *S. mansoni* worm (thin arrows) in the liver of a mouse surrounded by colonies of *Staphylococcus aureus* (thick arrow).

colonies. Larvae were observed in sections of the affected muscles, which were surrounded by an inflammatory infiltrate.

Our clinical and experimental data suggest an association between *T. canis* infection and tropical pyomyositis caused by *S. aureus*[20,36].

PYOGENIC LIVER ABSCESS AND TOXOCARIASIS

PEREIRA *et al.*[33] described observations of 22 cases of pyogenic liver abscess in children studied at autopsy (16 cases) or biopsy (6 cases), including 17 boys and 5 girls ranging in age from 1 to 13 years. Multiple abscesses in both lobes were found in 13 cases and a single abscess was found in the right lobe in 10 cases. All fragments examined histologically showed classical pyogenic inflammation without morphological evidence of amebiasis. In 6 cases there were granulomas similar to those caused by larva migrans visceralis (from *Toxocara* or other nematodes) in liver tissue not affected by the abscess. Nematode antigens in central areas of necrosis of granuloma in all 6 cases and fragments of a larva, possibly of *Toxocara*, were found on samples immunohistochemically stained with polyclonal anti-*Toxocara* antibodies. There were numerous eosinophils in abscesses with Charcot-Leyden cristals. Eosinophils were found frequently in portal triads far from the abscess wall. MOREIRA-SILVA & PEREIRA[28] expanded the data about this probable association. Also, RAYES *et al.*[34,35] showed that the serology for toxocariasis was positive in 10 of 16 patients with liver abscess against 4 of 32 matched controls without hepatic abscess, the difference being statistically significant. In an experimental study, mice were infected with eggs of *Toxocara* canis per os and a migrating larva was identified in the liver (Fig. 5). The authors suggest that toxocariasis is a predisposing cause for the development of pyogenic liver abscess.

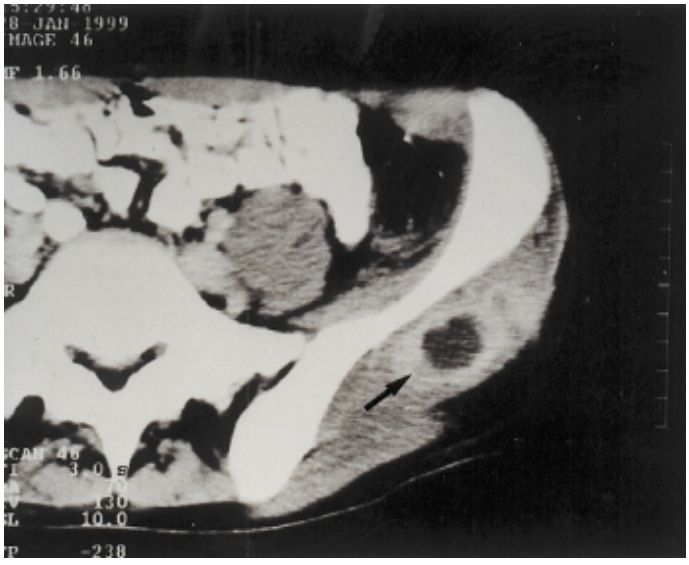

Fig. 4 - Computerized tomography showing an abscess (arrow) in the left gluteous muscle of a patient with tropical pyomyositis.

GRANULOMA, MATRIX COMPONENTS AND STAPHYLOCOCCI

There are data in experimental and human granulomatous disorders suggesting that during the formation of the typical hypersensitivity granuloma, a Th1 CD4+ T-cell profile predominates[14], while Th2 is associated with impaired granuloma formation and reduced resistance to intracellular pathogens. However, it is assumed that some granulomas may also be characterized by a Th2 pattern. For instance, T-cells surrounding granulomas arising in response to parasite ova chronically release Th2 cytokines while producing only small amounts of Th1 cytokines[26]. Therefore, differences in the type of cytokines locally produced may have

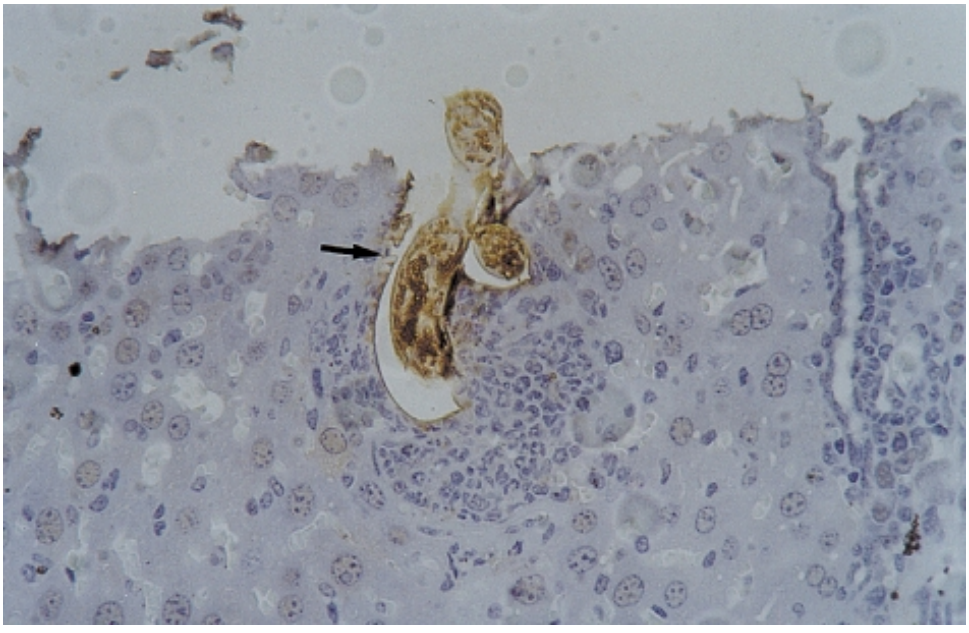

Fig. 5 - An inflammatory infiltrate around a larva in the liver of a mouse infected with *Toxocara canis* (immunohistochemical stain).

impact in determining the type of granuloma, its intensity and the extent of the central necrosis. In particular, it is believed that Th1 pattern provides maximum protective immunity to invading intracellular pathogens while Th2 cytokines are required for the infiltration and activation of soluble, toxic antigens, as in the case of schistosomiasis. The Th1/Th2 pattern is essential not only to determine the type of granulomatous response but also in the regulation of local fibrinogenetic processes. In granulomatous lesions a hyperplasia of fibroblasts may be observed and it is thought that a switch to a Th2 T-cell pattern with concomitant release of IL-4 (a cytokine, which is a chemotactic factor for fibroblasts and is able to stimulate the production of extracellular matrix proteins) may help the expansion of the mesenchymal cell population with increased deposition of extracellular matrix components in the environment surrounding granulomatous reactions[16].

It has been demonstrated that Gram-positive bacteria avidly bind to matrix-protein-coated surfaces. *S. aureus*, in particular, aggregates in the presence of fibronectin, laminin, and type IV collagen. As for collagen-associated proteoglycans, fibronectin is abundant during the more active stages of the schistosomal granuloma. This might suggest that compounds of the extracellular matrix of the granulomata of *S. mansoni* are involved in the adherence of *S. aureus*[27, 40].

CONCLUSION

Parasitic diseases may be associated with bacterial infections and be a predisposing factor for the development of pyogenic abscesses. Concomitant infection with migrating larvae of helminth or nematodes may switch the immune system from a T helper 1 response to a T helper 2 response, and, therefore, may also aggravate other infectious diseases such as tuberculosis, leprosy, leishmaniasis, AIDS, candidiasis and other superficial and deep mycosis, among others.

If our hypotheses are confirmed there will be a strong case in favor of the universal treatment of parasitic diseases (once a year?). Up to now the candidate drugs for treatment are albendazol, ivermectin and praziquantel. It would be desirable, though, to stimulate the pharmaceutical companies to develop new polivalent drugs capable of curing migrating larvae of different parasites.

RESUMO

Abscessos piogênicos e doenças parasitárias

As doenças parasitárias que durante a sua evolução no hospedeiro provocam mudança de uma resposta imune Th1 para uma resposta Th2 podem tornar-se prejudiciais ao hospedeiro, contribuindo para a formação de granulomas, eosinofilia, hiper-IgE, e suscetibilidade aumentada a infecções bacterianas e fúngicas. Demonstrou-se recentemente que animais e pacientes com esquistossomose aguda desenvolvem abscessos hepáticos piogênicos na presença de bacteriemia por *Staphylococcus aureus*. A associação da esquistossomose com bactérias do gênero *Salmonella* também encontra-se bem documentada. A infecção por *Toxocara* também parece predispor o hospedeiro a piomiosite tropical (abscesso muscular piogênico) e abscesso piogênico do fígado. Nos países tropicais essa poderia ser uma explicação para a maior morbidade das doenças bacterianas. Se a associação de doenças parasitárias e infecções bacterianas e fúngicas for confirmada sobrarão argumentos favoráveis ao tratamento universal das doenças parasitárias com o objetivo de prevenir ou diminuir a morbidade dessas infecções.

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